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Reply

Our study on nonfilling beats during balloon mitral valvuloplasty in humans failed to confirm negative diastolic left ventricular pressures previously observed by Nikolic (1) and others (2) in anesthetized open chest dogs using a mitral annulus occluder. This discrepancy is not explained by the methodologic issues raised by Nikolic.

In each patient, informed consent was obtained for balloon mitral valvuloplasty, which in both the United States and Europe has become standard therapy for mitral stenosis with suitable valve morphology. During balloon mitral valvuloplasty, left ventricular pressure is routinely monitored and insertion of a micromanometer-tipped catheter in the left ventricle does not add to patient risk or discomfort. In both laboratories in which measurements were performed, there was a long-standing experience with micromanometer-tipped catheters, which were calibrated against a mercury reference, checked against lumen pressure, eventually readjusted to match left ventricular end-diastolic pressure and checked for signal drift. Beats with a diastolic time interval <200 ms were excluded from analysis because a short diastolic time interval did not allow diastolic left ventricular pressure to decay to a value sufficiently close to the diastolic left ventricular asymptote. Short diastolic time intervals resulted from repetitive ventricular extrasystoles during balloon inflation. Balloon inflations without ventricular irritability resulted in an entire series of nonfilling beats suitable for analysis, and these patients therefore contributed more to overall data collection. Exclusion of Patients 6 and 12 because of higher diastolic left ventricular asymptote pressure and of slower left ventricular pressure decay seems inappropriate because of introduction of bias into the data collection and because these two patients had no outlying clinical characteristics. Moreover, even after their exclusion, an asymptote value of -0.5 ± 2 mm Hg still implies a positive transmural left ventricular diastolic asymptote pressure because of subatmospheric intrathoracic pressure during regular breathing. Finally, a hyperadrenergic state in animal preparations as a result of anesthesia and thoracotomy seems a well established fact even if its effect on heart rate is partially masked by previous sinoatrial node crush (1).

The discrepant results on the diastolic left ventricular asymptote pressure in our study in humans and data in dogs (1,2) are based on differences in underlying physiology. These differences could result not only from lower coronary perfusion during Inoue balloon inflation, as suggested by Nikolic, but also from altered extracellular matrix of the human heart and from inward motion into the left ventricular cavity of the balloon-occluded mitral valve as opposed to fixed position of the mitral annulus occluder (3) in animal experiments.

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